CLAIMS

1. A method for treating a disorder in which TNFα activity is detrimental comprising administering to a subject an effective amount of a TNFα inhibitor in a low dose therapy, such that the disorder is treated.

2. The method of claim 1, wherein the disorder is arthritis.

3. The method of claim 2, wherein the disorder is rheumatoid arthritis.

4. The method of claims 2 and 3, wherein symptoms selected from the group consisting of bone erosion, cartilage erosion, inflammation, and vascularity, are treated.

5. The method of any one of claims 1-3, wherein the TNFα inhibitor is an anti-TNFα antibody, or an antigen-binding portion thereof, or a TNFα fusion protein.

6. The method of claim 5, wherein the TNFα fusion protein is etanercept.

7. The method of claim 32, wherein D2E7 is administered in a low dose comprising 0.01 – 0.1 mg/kg.

8. A low dose method to alleviate symptoms associated with a disorder in which TNFα activity is detrimental, comprising administering a low dose of a TNFα inhibitor to a subject suffering from said disorder, such that the symptoms are treated.

9. The method of claim 8, wherein the disorder is arthritis.

10. The method of claim 9, wherein the disorder is rheumatoid arthritis.

11. The method of claims 9 and 10, wherein symptoms are selected from the group consisting of bone erosion, cartilage erosion, inflammation, and vascularity.

12. The method of any one of claims 8-10, wherein the TNFα inhibitor is an anti-TNFα antibody, or an antigen-binding portion thereof, or a TNFα fusion protein.

13. The method of claim 12, wherein the TNFα fusion protein is etanercept.

14. The method of claim 33, wherein D2E7 is administered in a low dose comprising 0.01 – 0.1 mg/kg.

15. A method for treating arthritis comprising administering to a subject an effective amount of a TNFα inhibitor in a low dose therapy, such that the arthritis is treated.

16. The method of claim 15, wherein the arthritis is rheumatoid arthritis.

17. The method of either claim 15 or 16, wherein arthritis is treated by alleviating symptoms selected from the group consisting of bone erosion, cartilage erosion, inflammation, and vascularity.

18 The method of claim 15 or 16 wherein the TNFα inhibitor is an anti-TNFα antibody, or an antigen-binding portion thereof, or a TNFα fusion protein.

19. The method of claim 18 wherein the TNFα fusion protein is etanercept.

20. The method of claim 34, wherein D2E7 is administered at a low dose comprising 0.01 – 0.1 mg/kg.

21. A low dose method for treating symptoms associated with arthritis comprising administering to a subject a low dose of an effective amount of a TNFα inhibitor, such that the symptoms are alleviated.

22. The method of claim 21, wherein the arthritis is rheumatoid arthritis.

23. The method of claims 21 and 22, wherein the symptoms are selected from the group consisting of bone erosion, cartilage erosion, inflammation, and vascularity.

24. The method of claim 23, wherein the symptoms are further selected from the group consisting of joint distortion, swelling, joint deformation, ankylosis on felxion, and severely impaired movement.

25. The method of claims 21 or 22, wherein the TNFα inhibitor is an anti-TNFα antibody, or an antigen-binding portion thereof, or a TNFα fusion protein.

26. The method of claim 25, wherein the TNFα inhibitor fusion protein is etanercept.

27. The method of claim 35, wherein D2E7 is administered at a low dose comprising 0.01 – 0.1 mg/kg.

28. A method of sequestering TNFα into complexes in a subject suffering from a disorder in which TNFα activity is detrimental, by administering a low dose of a TNFα inhibitor to the subject.

29. The method of claim 28, wherein the serum level of TNFα is higher than the serum level of TNFα in a subject not suffering from a disorder in which TNFα activity is detrimental.

30. The method of claim 28, wherein the anti-TNFα inhibitor is an anti-TNFα antibody, or an antigen-binding portion thereof, or a TNFα fusion protein.

31. The method of any one of claims 1, 8, or 15, wherein the TNFα inhibitor is administered with an additional therapeutic agent.

32. The method of claim 5, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is either infliximab or D2E7.

33. The method of claim 12, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is either infliximab or D2E7.

34. The method of claim 18, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is either infliximab or D2E7.

35. The method of claim 25, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is either infliximab or D2E7.

36. The method of claim 30, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is either infliximab or D2E7.

37. The method of claim 30, wherein the TNFα fusion protein is etanercept.

38. The method of claim 6, wherein etanercept is administered in a low dose comprising 0.01 - 1.0 mg/kg.

39. The method of claim 32, wherein infliximab is administered in a low dose comprising 0.01 – 0.5 mg/kg.

40. The method of claim 5, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is human.

41. The method of claim 40, wherein the anti-TNFα antibody, or antigen-binding portion thereof, dissociates from human TNFα with a K_d of 1 x 10^{-8} M or less and a K_{off} rate constant of 1 x 10^{-3} s^{-1} or less, both determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1 x 10^{-7} M or less.

42. A low dose method for treating rheumatoid arthritis in which TNFα activity is detrimental comprising administering to a subject a low dose of a human TNFα antibody, or an antigen-binding portion thereof, such that the disorder is treated.

43. The method of claim 36, wherein symptoms selected from the group consisting of bone erosion, cartilage erosion, inflammation, and vascularity, are treated.

44. The method of claim 36, wherein the anti-TNFα antibody, or antigen-binding portion thereof, dissociates from human TNFα with a K_d of 1 x 10^{-8} M or less and a K_{off} rate constant of 1 x 10^{-3} s^{-1} or less, both determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1 x 10^{-7} M or less.

45. The method of claim 36, wherein the anti-TNFα antibody, or antigen-binding portion thereof, is D2E7.

46. The method of claim 36, wherein the amount of antibody, or antigen-binding portion thereof, administered to the subject comprises 0.01 – 0.1 mg/kg.

47. The method of any one of claims 1, 8, 15, or 21, wherein the amount of anti-TNFα antibody, or antigen-binding portion thereof, administered to the subject comprises 0.01 – 0.1 mg/kg.

48. A low dose method of improving symptoms in the joints of a subject having arthritis comprising administering to the subject a low dose of a human anti-TNFα antibody, or antigen-binding portion thereof, comprising 0.01-0.1 mg/kg such that at least one symptom selected from the group consisting of inflammation, cartilage erosion, bone erosion, and vascularity is improved.

49. The method of claim 44, wherein the anti-TNFα antibody, or an antigen-binding portion thereof, is D2E7.